# MODELING LONGITUDINAL RELATIONS BETWEEN STRESS AND DEPRESSIVE SYMPTOMS: COPING AS A MEDIATOR

By

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# CHAPTER I

## INTRODUCTION

Psychosocial stress has a significant and lasting impact on children. Longitudinal research has revealed a significant prospective relation between stress and psychopathology in children and adolescents (Hammen, & Goodman-Brown, 1990; Hilsman, & Garber, 1995; Rudolph, Lambert, Clark, & Kurlakowsky, 2001). Specifically, a positive association between stress and higher levels of depressive symptoms has been reported in both community and outpatient samples of children and adolescents (Barrera et al., 2002; Barrett, & Heubeck, 2000; Rudolph et al., 2000; Sandler, Reynolds, Kliewer, & Ramirez, 1992; Wadsworth, & Compas, 2002). Chronic stress can disrupt developmental processes, and researchers have emphasized the need for increased exploration of the mechanisms that underlie the relation between stress and psychopathology in children and adolescents (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001). Understanding the processes that contribute to these relations will facilitate the development of more effective interventions for preventing and treating child psychopathology.

One mechanism that affects the relation between stress and psychological symptoms is the process by which children respond and adapt to stressors (Compas, Malcarne, & Fondarcaro, 1988; Compas et al., 2001). Examining children's coping strategies in response to stress can potentially reveal why some children are vulnerable to developing psychopathology as well as identify ways to promote adaptive functioning

and positive growth in children exposed to significant adversity. Although links among stress, coping, and psychopathology have been found (e.g., Sandler, Tein, & West 1994; Wadsworth, Raviv, Compas, & Connor-Smith, 2005), the extent to which specific types of coping strategies longitudinally mediate the relation between stress and depressive symptoms remains unclear. The purpose of this prospective study was to examine the relations among stressful life events, coping, and depressive symptoms in a sample of children at varied risk for depression.

*Conceptualization of Coping*

Coping is broadly defined as the "conscious volitional efforts to regulate emotion, cognition, behavior, physiology, and the environment in response to stressful events or circumstances" (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001, pg. 89). Although coping responses may be somewhat stable due to individual differences, coping generally is regarded to be a process, rather than a trait, which can be influenced by the context of the stressors. For instance, studies have shown that characteristics of the stressor such as its controllability can impact patterns of coping and their effectiveness (Compas, Connor, Harding, Saltzman, & Wadsworth, 1999). Thus, some aspects of coping are state-like and some are trait-like; stability over time may reflect an individual's attempt to cope repeatedly with the same chronic stressor (Wadsworth, & Berger, 2006).

The current study was guided by the model of stress responses proposed by Connor-Smith, Compas, Wadsworth, Thomsen and Saltzman (2000) that highlights both voluntary and involuntary responses. Voluntary coping responses can be further

categorized as either engagement with or disengagement from a stressful life event and one's emotional reactions to that stressor (Compas et al., 2001; Connor-Smith, et al., 2000). Confirmatory factor analyses have revealed a model of coping in children and adolescents that includes primary control engagement coping, which are attempts to directly change the situation or one's emotional reaction (e.g., problem solving, emotional expression), secondary control engagement coping, which are attempts to adapt to the situation by regulating attention and cognition (e.g., acceptance, cognitive restructuring, positive thinking, distraction), and disengagement coping (e.g., avoidance, denial, wishful thinking), which involves withdrawing from the source of stress and one's emotions. Evidence across samples and types of stressors indicates that coping strategies that involve engagement with the stressor or one's emotional reaction to the stressor (primary and secondary control coping, respectively) are associated with better outcomes, whereas strategies that involve disengagement from the stressor are associated with more symptoms and lower competence (Compas et al., 2001). Using this framework, the present study investigated the relations among these three voluntary coping efforts, stressful events, and symptoms of psychopathology in children and adolescents at varied risk for depression.

*Coping as a Mediator between Stress and Psychopathology*

Mediation assumes that coping may be affected by stressful conditions, and thus is not a static characteristic. That is, stress may determine the coping strategies that are enacted, and the use of those specific strategies then contributes to the link between stress and psychopathology (Wadsworth et al., 2005). Stress can impact the brain and impede

an individual's ability to cope effectively (Compas, 2006; Wellman, 2001). For example, children exposed to higher levels of stress are less likely to utilize constructive, cognitively demanding coping methods such as problem-solving and cognitive restructuring (Valiente, Fabes, Eisenberg, & Spinrad, 2004). Moreover, children exposed to high levels of stress are under more cognitive load and therefore are less likely to use coping strategies that require a greater cognitive capacity (Matthews, & Wells, 1996).

Cross-sectional investigations of children have shown that coping becomes less effective under conditions of high stress (Jaser et al., 2008; Jaser et al., 2005; Wadsworth, Reickmann, Benson, 2004). Specifically, the use of primary control and secondary control coping decreases as stress increases (Wadsworth & Compas, 2002). Additionally, among offspring of depressed parents, high stress has been found to be associated with higher levels of arousal, intrusive thoughts and rumination, and less secondary control coping (Langrock, Compas, Keller, & Merchant, 2002). Thus, the strong inverse relation between involuntary stress reactivity and adaptive coping suggests that the ability to use advanced cognitive strategies, such as problem solving, may be compromised by elevated stress (Compas, 2006; Valiente et al., 2004).

Several studies have specifically examined coping as a mediator. In a cross-sectional investigation of offspring of depressed parents, secondary control coping was found to partially mediate the relation between parent-child interaction stress and children's symptoms of anxiety and depression as well as the relations between peer and family stressors and internalizing symptoms (Jaser et al., 2005). Moreover, different coping strategies used to deal with different types of stressors (e.g., controllable and uncontrollable) are then related to adjustment. In particular, secondary control coping

strategies in response to family stress and primary control coping in response to peer stress have been found to be associated with fewer symptoms of anxiety/depression and aggression (Jaser et al., 2007). In a cross-sectional study of children exposed to poverty-related stressors, secondary control coping was found to mediate the relation between stressors and symptoms (Wadsworth, Raviv, Compas, & Connor-Smith, 2005). Additionally, Wadsworth and colleagues found that in adolescents, coping mediated the stress-symptom relation, whereas in adults coping responses moderated this relation.

Prospective studies also have found some evidence of coping as a mediator. Dempsey (2002) reported that negative coping mediated the relation between exposure to community violence and depression and anxiety symptoms in a sample of inner-city African American adolescents. In a study examining children of divorce, Sandler and colleagues (1994) reported that active coping and distraction predicted lower internalizing symptoms, whereas support coping, which involves seeking out others for emotional support or to assist with problem solving, predicted higher levels of depressive symptoms. Investigations of children exposed to marital conflict (Shelton, & Harold, 2007) and adolescent girls in juvenile justice (Goodkind, Ruffolo, Bybee, & Sarri, 2009) have revealed that the long-term effects of stressors on children's symptoms of anxiety and depression were mediated by the use of maladaptive coping responses such as withdrawal, venting of emotions, and acting out coping. Sawyer, Pfieffer and Spence (2009) examined the longitudinal relations among coping, optimistic thinking style, and depressive symptoms in a large normative sample, and found that negative coping styles acted as a moderator, such that negative coping strategies interacted with stress levels to predict higher levels of depressive symptoms. Thus, results of these longitudinal studies

are consistent with a model that high levels of stress may impede children's ability to use cognitively demanding coping strategies, which in turn contribute to increased risk of psychopathology.

*The Present Study*

The current study builds upon existing research on coping in children and adolescents in several ways. First, most studies examining coping as mediator have been cross-sectional. Empirical tests of mediation using a cross-sectional design can be potentially misleading (see Cole, & Maxwell, 2003). Second, the conceptualization and operationalization of coping has varied in this literature, thereby making it difficult to compare findings across studies. Investigators have called for theoretically-driven, incremental research using comparable measures to examine the role of specific coping strategies in relation to stress and symptoms (Grant et al., 2006). Following this recommendation, the current study used the model of coping developed by Connor-Smith and colleagues (2000), which has been examined in a number of recent cross-sectional investigations of coping as a mediator.

Third, several of the studies examining the relations among stress, coping responses, and depressive symptoms have used high-risk samples of offspring of depressed parents (Langrock et al., 2002; Jaser et al., 2005) and have assessed how children cope with the uncontrollable stressor of having a depressed parent. These studies have found that secondary control coping mediates the relation between the stressor of parental depression and children's depressive symptoms (Jaser et al., 2005; Langrock et al., 2002). The present sample also was comprised of offspring of depressed and

nondepressed parents, but coping was assessed with regard to more general stressors rather than the specific stress of having a depressed parent. A strength of such a coping measure is that it assesses coping strategies in response to both controllable and uncontrollable events that occur within a given time period.

Finally, children in the current study varied in their risk for psychopathology. This sample was used because offspring of depressed parents are at heightened risk for developing both internalizing and externalizing problems as compared to offspring of non-depressed parents (Beardslee, Versage, & Gladstone, 1998; Hammen, & Brennan, 2003; Lieb, Isensee, Hofler, Pfister, & Wittchen 2002). One mechanism by which risk is conferred is through increased exposure to chronic stressors associated with parental depression (Goodman, & Gotlib, 1999); disrupted parenting associated with symptoms of depression (i.e. sadness, irritability) contributes to stressful family environments (Hammen, 2003). Life with a depressed parent often is characterized by negative and unpredictable parent behavior (e.g. hostility and intrusiveness) toward the child (Lovejoy, Graczyk, O'Hare, & Neuman, 2000), greater parental withdrawal (Langrock, Compas, Keller, Merchant, 2002), and high levels of marital discord (Cummings, Keller, & Davies, 2005). Additionally, children of depressed parents may have poor interpersonal functioning, which then may contribute to the number and types of stressors they experience outside of the home (Adrien, & Hammen, 1993).

Finally, offspring of depressed parents tend to engage in less effective coping behaviors (Garber, Braafladt, & Weiss, 1995; Jaser et al., 2005). Thus. offspring of depressed parents not only are exposed to high levels of controllable and uncontrollable stress, but they also tend to lack skills for coping with these stressors. Such a high-risk

research design provides increased variability and power to detect indirect effects among the three constructs of interest: stress, coping, and depressive symptoms. In summary, the current study was a multiple assessment, longitudinal study using a theoretical model of coping to examine the meditational relations among stress, coping, and children's depressive symptoms in a sample that varied in risk for depression.

The present investigation compared three models involving primary control, secondary control, and disengagement coping as mediators of the relation between stressful life events and depressive symptoms in youth. The present study tested the hypothesis that there would be a positive relation between prior stressful life events and subsequent depressive symptoms in children at the next time point. Moreover, we expected this relation to be indirect and partially mediated by coping strategies. We further hypothesized that primary control and secondary control coping would be negatively associated with stressful life events and negatively associated with depressive symptoms. Conversely, we expected that disengagement coping would be positively associated with stressful life events and positively associated with depressive symptoms. Additionally, the relation between stress and depression is likely to be bidirectional; that is, stress and symptoms reciprocally predict each other, and may be mediated by coping, Thus, an important goal of the present study was to link children's coping responses to the trajectory of their depressive symptoms over time.

# CHAPTER II

# METHOD

## *Participants*

Participants were 227 dyads of one parent and one child per family. The high-risk group consisted of 129 families in which a parent was receiving treatment for a current Major Depressive Disorder (MDD) as defined in the *Diagnostic and Statistical Manual of Mental Disorders* (4th edition; American Psychiatric Association, 1994), and scored 14 or greater on the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967). Exclusion criteria included a lifetime diagnosis of any psychotic or paranoid disorder, organic brain syndrome, mental retardation, or bipolar I or II, current or primary diagnosis of substance abuse or dependence, obsessive-compulsive disorder, eating disorder, certain personality disorders (antisocial, borderline, schizotypal), or unwillingness to participate in treatment for depression.

The comparison group (low risk) included 98 families with parents who were lifetime-free of mood disorders, psychotic disorders, organic brain syndromes, or personality disorders, and during the child's life free of adjustment disorders, anxiety disorders, substance abuse/dependence, psychotherapy longer than two months or eight sessions, and psychotropic medication use.

Child participants were between 7 and 17 years old (Mean = 12.13, *SD* = 2.31). Exclusion criteria included a developmental disability or significant chronic medical conditions. For the nondepressed families, the enrolled child was selected to be similar in

age and gender to a high-risk child. The overall sample was 54.6% female, 69.6% Caucasian, 21.6% African-American, 1% Asian, and 6.9% multi-racial. High- and low-risk children did not differ significantly in children's age, gender, ethnicity/race, or parents' age or gender (see Table 1); the groups were significantly different on parent education, which therefore was controlled in all analyses.

*Procedure*

Depressed parents were recruited from clinics as they first presented for treatment for depression. These parents received standard, evidence-based treatments including medications and/or cognitive behavioral therapy from experienced psychiatrists, psychologists, social workers, and psychiatric nurses. Recruitment of comparison families involved print and radio advertisements, and coordination with local schools, health maintenance organizations, and community agencies. These parents were initially screened over the telephone, and if eligible, then were scheduled for a clinical evaluation to further assess inclusion and exclusion criteria.

For the high-risk group, child assessments occurred at the beginning of the parents' treatment and were conducted by different evaluators than those doing the parent assessments. Follow-up evaluations of children were conducted at parents' mid- (2 months) and post-treatment (4 months) and then at 6-month intervals after that (i.e., 10, 16, and 22 months post baseline). This paper reports results from the T1 (baseline), T3 (4 month), T5 (16 month) and T6 (22 month) follow-up assessments. These assessment points were used in the present analysis because measures of stressful life events, coping, and depressive symptoms were obtained at these four time points. For the low-risk group,

children first were assessed within two weeks after the parent evaluation and then also followed at these multiple points across time. At the baseline interview, the IRB-approved written informed parent consent and child assent were obtained from all participants. Only measures used in the current study are described here.

*Measures*

Parents' Psychopathology

*The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I;* First et al., 1997) was used to evaluate parents' psychopathology. A randomly selected subset of taped interviews was used to assess inter-rater reliability, yielding kappa coefficients $\geq$ .80.

*Hamilton Rating Scale for Depression* (HRSD; Hamilton, 1967) is an interview-based measure of the severity of depression. The 17-item version used here yields scores ranging from 0 to 52; higher scores indicate greater severity. The HRSD has high inter-rater reliability (i.e., $\geq$ .84). Intra-class correlation in this study was .96.

*Beck Depression Inventory, Second Edition (BDI-II;* Beck et al., 1996; Beck, Steer, & Garbin, 1988) is a self-report inventory with 21 items rated on a four-point scale ranging from 0 (absence of symptoms) to 3 (most intense level of the symptom). Scores can range from 0 to 63, with higher scores indicating more depression. The BDI-II is consistent with DSM-IV criteria for depressive disorders and has good psychometric properties (Beck et al., 1996). Coefficient alpha of the BDI-II in this sample was $\geq$ .93 at all time points.

Children's Depressive Symptoms, Coping, Stressful Life Events

The *Children's Depression Inventory* (CDI; Kovacs, 1992) measures self-reported symptoms of depression in children. Each of the 27 items lists three statements in order of increasing severity. Total scores can range from 0 to 54, with higher scores indicating more depression. Internal consistency, test-retest reliability, and convergent validity have been well documented for the CDI (Kovacs, 1992). Coefficient alpha for the CDI in this sample was $\geq$ .84 at all time points.

The *COPE Inventory* (Carver, Scheier, Weintraub, 1989) is a multidimensional coping inventory representing an integration of various theoretical models. On the dispositional COPE, individuals rate on a four-point Likert scale (1: *Usually do not do this at all*; 4: *Usually do this a lot*) the extent to which they generally use each of 60 coping strategies. Internal consistency, test-retest reliability, and convergent and discriminant validity have been well documented for the COPE (Carver et al., 1989).

The 60 items of the COPE can be divided into 15 subscales measuring conceptually and empirically distinct aspects of coping. These subscales have been shown to correlate with measures of primary control, secondary control, and disengagement coping as defined in the model proposed by Connor-Smith and colleagues (2000). Based on convergent and discriminant validity correlations, Connor-Smith et al. (2000) created coping composites from the COPE subscales to represent a three-factor model comprised of primary, secondary, and disengagement coping. The COPE subscales included in the primary control coping composite were Planning, Seeking Social Support for Instrumental Reasons, Active Coping, and Seeking Social Support for Emotional Reasons. The secondary control coping composite included the Acceptance

and Positive Reinterpretation and Growth subscales; the disengagement composite included Behavioral Disengagement, Denial, and Mental Disengagement subscales. As recommended by Connor-Smith and colleagues, we computed factor scores for coping as proportions of the total score for all responses (i.e., sum of scores on primary control items/sum of all items) to control for overall responding bias. In the current sample, internal consistency across all time points was adequate (primary control coping: $\alpha \geq .88$; secondary control coping: $\alpha \geq .86$; disengagement coping: $\alpha \geq .72$).

The *Life Events Questionnaire* (LEQ; Johnson, & McCutcheon, 1980) measures self-reported stressful life events in children. The LEQ includes 50 stressful life events, such as: *A family member or close relative died; You got into serious trouble at school*. Scores can range from 0 to 50, with higher scores indicating more stressful events during the specified period. At baseline, children reported stressful events that had occurred in the last year, and for all subsequent time points children reported stressful life events that had occurred since the previous assessment. Versions of the LEQ have been widely used in studies of adverse events among children and adolescents with various psychiatric disorders and has good psychometric properties (Johnson, & McCutcheon, 1980).

# CHAPTER III

# RESULTS

## *Descriptive Analyses*

Table 1 presents the means, standard deviations, and correlations of the study variables. Intercorrelations among the measured variables from T1-T6 are presented in Table 2. Child age was not significantly correlated with primary or secondary coping, but was positively correlated with stressful life events (*rs* = 0.10 to 0.20) and depressive symptoms (*rs* = 0.02 to 0.19), and negatively correlated with disengagement coping (*rs* = 0.01 to -0.14). Child gender was not significantly correlated with primary, secondary, disengagement coping or depressive symptoms across time points, but was negatively correlated with stressful life events (*rs* = -0.09 to -0.19), indicating that girls reported more stressful events than boys. Parental depressive symptoms were not significantly correlated with primary control coping, but were negatively correlated with children's secondary control coping (*rs* = -0.04 to -0.18), and positively correlated with disengagement coping (*rs* = 0.15 to 0.17), stressful events (*rs* = 0.21 to 0.40), and children's depressive symptoms (*rs* = -0.02 to 0.24).

Correlations among the proposed indicators were moderate and often significant. Stressful life events were positively correlated with disengagement coping (*rs* =0.14 to 0.30) and negatively correlated with primary control coping and secondary control coping at the next time point (*rs* = -0.03 to -0.24). Primary and secondary coping strategies were negatively correlated with depressive symptoms at the subsequent time point (*rs* = -0.16

to -0.35), whereas disengagement coping was positively correlated with depressive symptoms at the subsequent time point (*rs* = 0.30 to 0.43). These associations provide evidence of the relations among the variables that comprise the meditational model.

*Model Testing Approach*

Structural equation modeling (SEM) using AMOS 17.0 (Arbuckle, 2008) was used to test for longitudinal cross-lagged effects. This method was chosen over regression modeling because of its ability to test models with multiple dependent variables, the ability to model mediating variables rather than being restricted to an additive model as in regression, and the ability to model error terms. Full information maximum likelihood (FIML) estimation was used, which handles missing data. Three auto-regressive cross-lag SEMs were fit, each modeling the relations among study variables using primary, secondary, and disengagement coping. The models tested examined whether stressful life events directly predicted depressive symptoms and indirectly predicted depressive symptoms through the mediator, primary control coping, secondary control coping, or disengagement coping (see Figure 4). All relevant auto-regressive paths were included, following guidelines by Cole and Maxwell (2003) for testing longitudinal mediation. According to Cole and Maxwell (2003), if typical cross-sectional mediation procedures are applied without controlling for prior levels of all the model variables, it is assumed that prior levels of the variables are unrelated to subsequent levels, which is highly unlikely in developmental psychopathology research. In the present analysis, all auto-regressive paths were included to reduce this potential bias and provided a stringent test of longitudinal direct and indirect effects in each model. Models also controlled for

concurrent correlations among variables. Age, gender, and parent depressive symptoms (BDI) at Times 1, 3, 5, and 6 were entered into the model and controlled for at each time point. Additionally, it should be noted that T1 CDI scores strongly predicted T5 CDI scores. When an auto-regressive path from T1 to T5 was included in the model, however, the relation between T3 and T5 CDI scores was no longer significant.

The significance of the standardized path coefficients was determined by comparing the *t* ratio with a critical *t*(0.05) of 1.96. Model fit was assessed using several indicators and thresholds recommended by Hu and Bentler (1998, 1999), including the chi-square statistic, the comparative fit index (CFI), the incremental fit index (IFI) and the root-mean-square error of approximation (RMSEA). Models that provided a good fit to the data had non-significant chi-square values, CFI greater than .95, IFI greater than .95, and RMSEA of less than .06.

The significance of the indirect effect between stressful life events, coping, and depressive symptoms was calculated using a bias-corrected bootstrap procedures. The bootstrap procedure was used rather than the Sobel test, which has been shown to be low in power and high in type I error rate (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). In many cases, statistical testing assumes that the estimate of the indirect effect follows a normal distribution; however, this assumption often is inappropriate and likely is a reason that the Sobel test does not perform as well.

In the bootstrap procedure, an original data set is used to create a large number of randomly drawn additional data sets of the same size. Over many bootstrap re-samples, an empirical approximation of the sampling distribution of the statistic can be generated and used for hypothesis testing. The bootstrap procedure conducted in the present

analysis resulted in a 95% bias-corrected confidence interval of the indirect effect of stressful events on depressive symptoms. It should be noted that the bootstrap was conducted after imputing missing data using the linear interpolation procedure provided by SPSS 17.0.

*Primary Control Coping*

Model testing began by examining a model that included stressful life events, depressive symptoms, and primary control coping measured at all time points. The model included direct paths between stressful life events and depressive symptoms as well as indirect paths to and from the mediator, primary coping, in addition to autoregressive and reciprocal paths between all variables. This model provided a good fit to the sample data, $\chi^2$ (14, 227) = 21.379, $p$ = .09; IFI = 0.99, CFI = 0.99, RMSEA = 0.05.

*Direct effects.* The direct effects between stressful life events and depressive symptoms are presented in Table 3. Stressful events that occurred from T1 to T3 significantly predicted higher levels of depressive symptoms at T3 (β = .41, $p$ < .001), and marginally predicted higher levels of depressive symptoms at T5 (β = .14, $p$ = .09). Stressful life events from T3 to T5 significantly predicted higher levels of depressive symptoms at T5 (β = .28, $p$ < .001); the prediction from stressful life events from T5 to T6 to depressive symptoms at T6 indicated a nonsignificant trend (β = .12, $p$ = .09). Examining the reverse direction of effects, depressive symptoms at T1 significantly predicted more stressful life events from T3 to T5 (β = .20, $p$ < .01). The significance of these paths suggests that there was a positive direct effect between stressful life events and children's reported depressive symptoms, such that higher levels of prior stress

predicted increases in children's reported depressive symptoms, and at T1 higher levels of depressive symptoms predicted more subsequent stressful life events.

*Indirect Effects.* Inspection of the standardized path coefficients indicated that stressful life events between T3 to T5 significantly predicted less primary control coping at T5 ($\beta = -.20$, $p < .01$); a negative path coefficient revealed that lower levels of primary control coping at T5 significantly predicted higher levels of depressive symptoms at T6 ($\beta = -.15$, $p < .01$) (see Figure 1). Confidence intervals of the indirect effects of stressful events on depressive symptoms (95% CI: 0.20 – 0.55) based on 500 bootstrap samples did not include zero. Taken together, these results suggest that from T5 to T6 the relation between stressful life events and depressive symptoms was mediated by primary control coping. That is, stressful events predicted lower levels of primary control coping, which in turn predicted higher levels of depressive symptoms.

Examining the reverse direction of effects, that is, the indirect paths from depressive symptoms to stressful events through the mediator, primary coping, indicated that depressive symptoms at Times 3 and 5 predicted subsequent primary control coping at T5 ($\beta = .173$, $p < .05$) and T6 ($\beta = -.198$, $p < .01$), respectively. At T1, greater use of primary control coping predicted fewer stressful events from T3 to T5 ($\beta = -.138$, $p < .05$).

*Secondary Control Coping*

The second model included direct paths between stressful life events and depressive symptoms as well as indirect paths to and from the mediator, secondary control coping, in addition to autoregressive and reciprocal paths between all variables.

The model provided a good fit to the sample data, $\chi^2$ (14) = 24.172, $p$ = .044; IFI = 0.99, CFI = 0.99, RMSEA = 0.06.

*Direct effects.* The direct effects presented in Table 4 between stressful life events and depressive symptoms were generally similar to those in the first model, such that stressful events that occurred from T1 to T3 significantly predicted higher levels of depressive symptoms at T3 (β = .39, $p$ < .001) and at T5 (β = .16, $p$ < .05). Additionally, stress from T3 to T5 significantly predicted depressive symptoms at T5 (β = .26, $p$ < .001). The paths from depressive symptoms to stressful life events revealed that depressive symptoms at T1 significantly predicted more stressful events from T3 to T5 (β = .25, $p$ < .001).

*Indirect effects.* In contrast to the primary control coping model, evidence for an indirect effect between stressful events and depressive symptoms through secondary control coping was not found (see Figure 2). Evidence for a indirect effect between depressive symptoms and stressful events through secondary coping was also not found. There was a significant, negative path coefficient between depressive symptoms at T1 and secondary control coping at T3 (β = -.178, $p$ < .01). All other paths in the model were not significant.

*Disengagement Coping*

The third model included direct paths between stressful life events and depressive symptoms as well as indirect paths to and from the mediator, disengagement coping, in addition to autoregressive and reciprocal paths between all variables. The model provided

a good fit to the sample data, $\chi^2$ (14) = 27.241, $p$ = .02; IFI = 0.99, CFI = 0.99, RMSEA = 0.06.

*Direct effects.* The pattern of direct effects between stress and depressive symptoms was very similar to the pattern observed in the primary and secondary control models (see Table 5). Stressful events that occurred from T1 to T3 predicted higher levels of depressive symptoms at T3 (β = .40, $p$ < .01) and stressful events from T3 to T5 predicted depressive symptoms at T5 (β = .28, $p$ < .01). Examining the reverse direction of effects indicated that only depressive symptoms at T1 predicted a greater number of stressful life events from T3 to T5 (β = .19, $p$ < .01). The significance of these paths suggested that there was a positive direct effect between stressful life events and children's depressive symptoms between T1 and T5, such that children's depressive symptoms predicted more stressful life events, which in turn predicted higher levels depressive symptoms.

*Indirect effects.* Inspection of the standardized path coefficients indicated that stressful life events from T3 to T5 were significantly associated with greater use of disengagement coping at T5 (β = .13, $p$ < .05), and greater disengagement coping at T5 significantly predicted higher levels of depressive symptoms at T6 (β = .17, $p$ < .01) (see Figure 3). Confidence intervals of the indirect effects of stressful events on depressive symptoms (95% CI: 0.18 – 0.55) based on 500 bootstrap samples did not include zero. The indirect effects suggest that stressful events from T3 to T5 predicted higher levels of disengagement coping, which in turn predicted higher levels of depressive symptoms at T6. Thus the relation between stressful life events and depressive symptoms was partially mediated by disengagement coping.

Examining the reverse direction of effects, or the indirect paths from depressive symptoms to stressful events through disengagement, revealed that higher levels of depressive symptoms at T1 predicted greater use of disengagement coping at T3 (β = .186, $p < .01$), and higher levels of depressive symptoms at T5 predicted greater use of disengagement coping at T6 (β = .12, < .05). Depressive symptoms at T3 predicted lower levels of disengagement coping at T5 (β = -.19, $p < .01$). At T1, disengagement coping predicted a greater number of stressful life events from T3 to T5 (β = .17, $p < .01$).

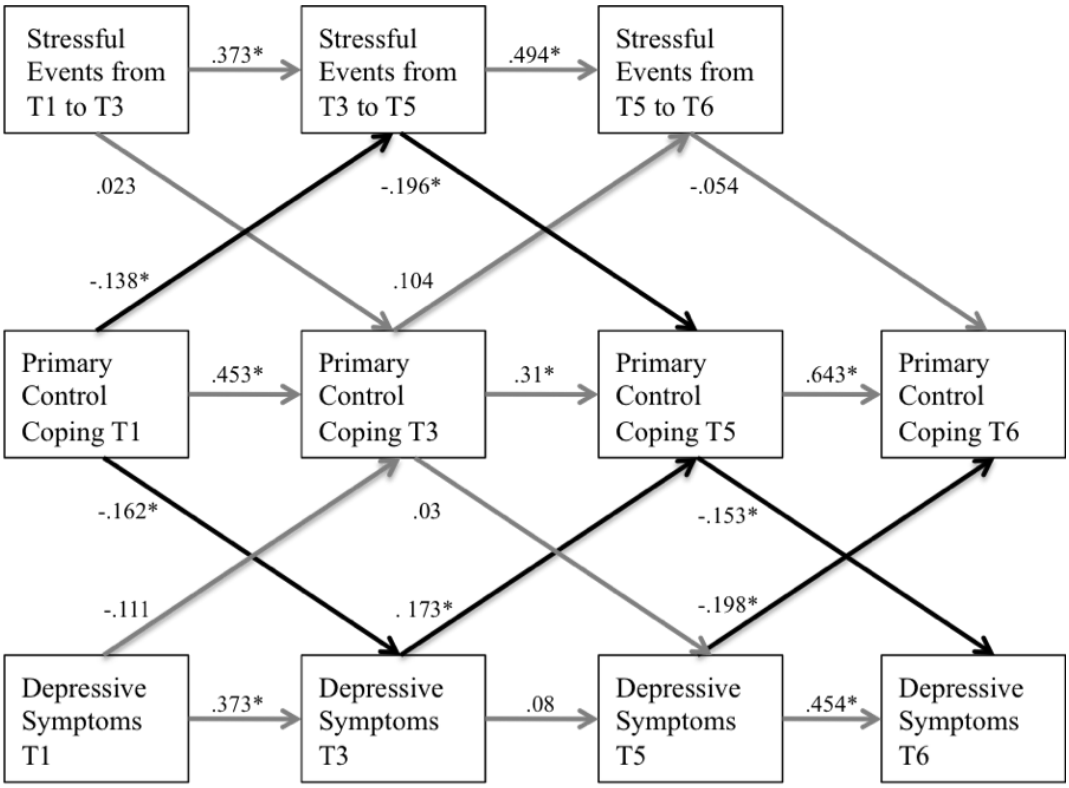


*Figure 1.* Primary control coping model: mediating effects. Direct paths between Depressive Sxs and Stressful Events, nonadjacent autoregressive paths, and covariances between errors within each time point were estimated but not depicted. Covariates included age, gender, and T1, T3, T5 and T6 Parent BDI (Beck Depression Inventory) scores which predicted all variables in model. * $p < .05$

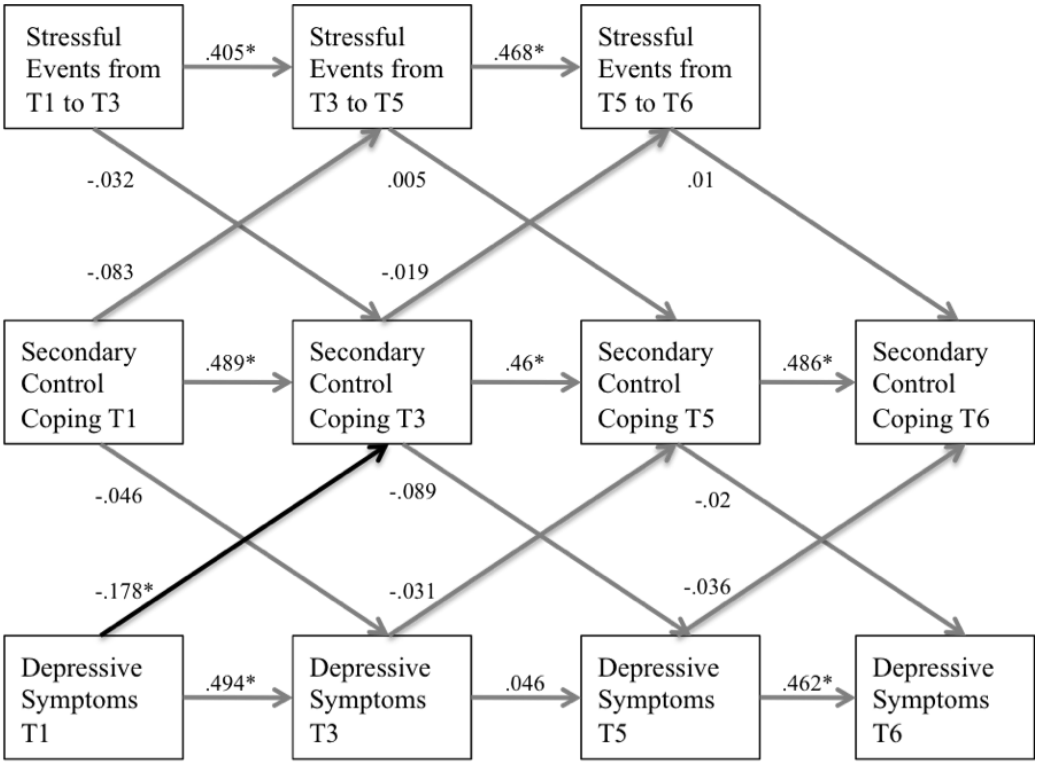


*Figure 2.* Secondary control coping model: mediating effects. Direct paths between Depressive Sxs and Stressful Events, nonadjacent autoregressive paths, and covariances between errors within each time point were estimated but not depicted. Covariates included age, gender, and T1, T3, T5 and T6 Parent BDI (Beck Depression Inventory) scores which predicted all variables in model. * $p < .05$

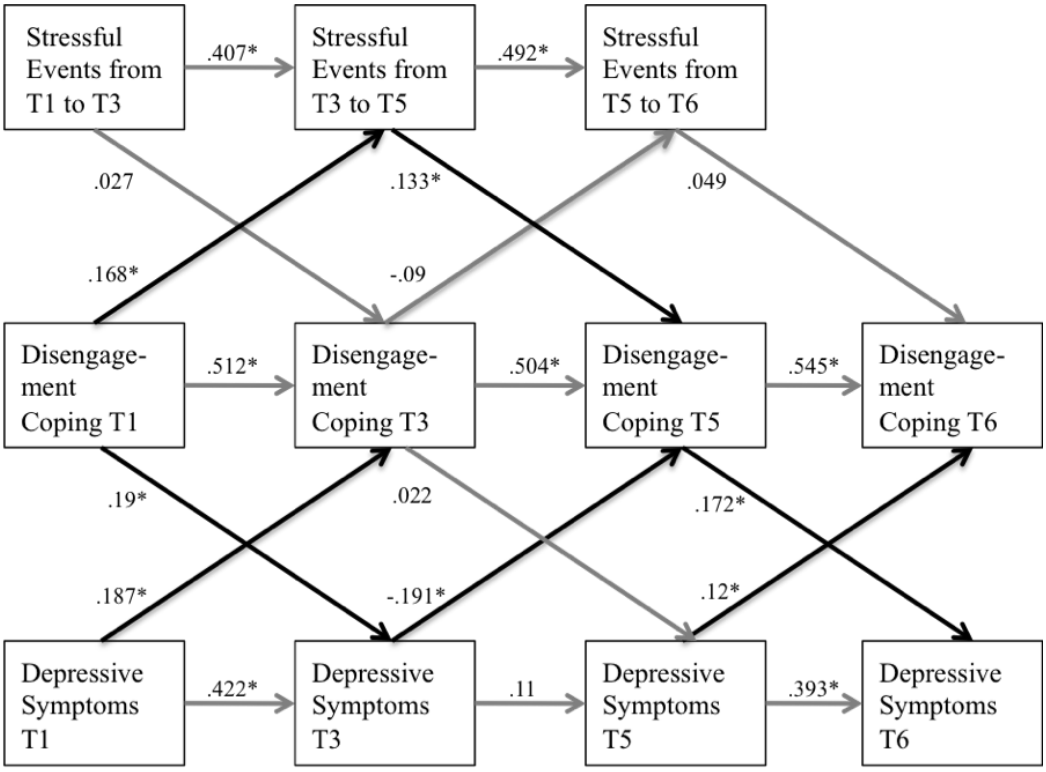


*Figure 3.* Disengagement coping model: mediating effects. Direct paths between Depressive Sxs and Stressful Events, nonadjacent autoregressive paths, and covariances between errors within each time point were estimated but not depicted. Covariates included age, gender, and T1, T3, T5 and T6 Parent BDI (Beck Depression Inventory) scores which predicted all variables in model. * $p < .05$

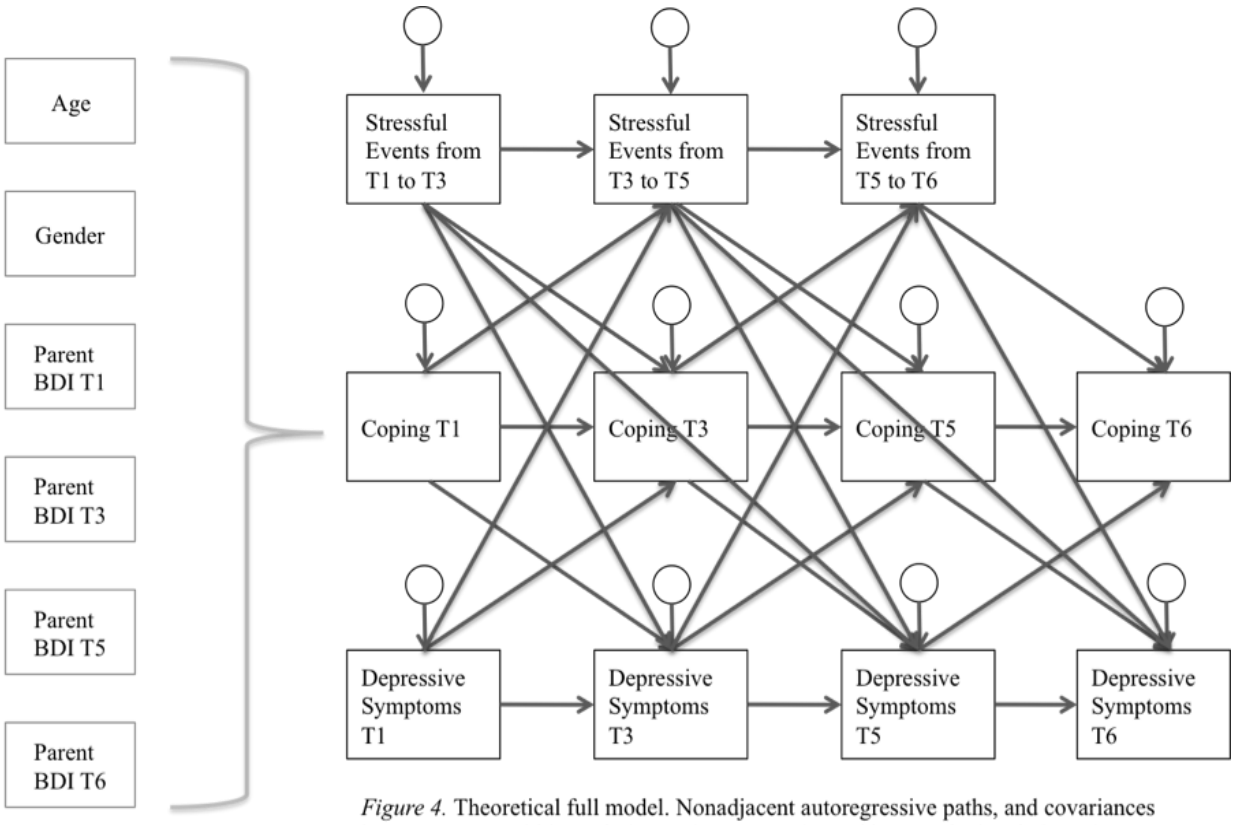


*Figure 4.* Theoretical full model. Nonadjacent autoregressive paths, and covariances between errors within each time point were estimated but not depicted. Covariates included age, gender, and T1, T3, T5 and T6 Parent BDI (Beck Depression Inventory) scores which predicted all variables in model.

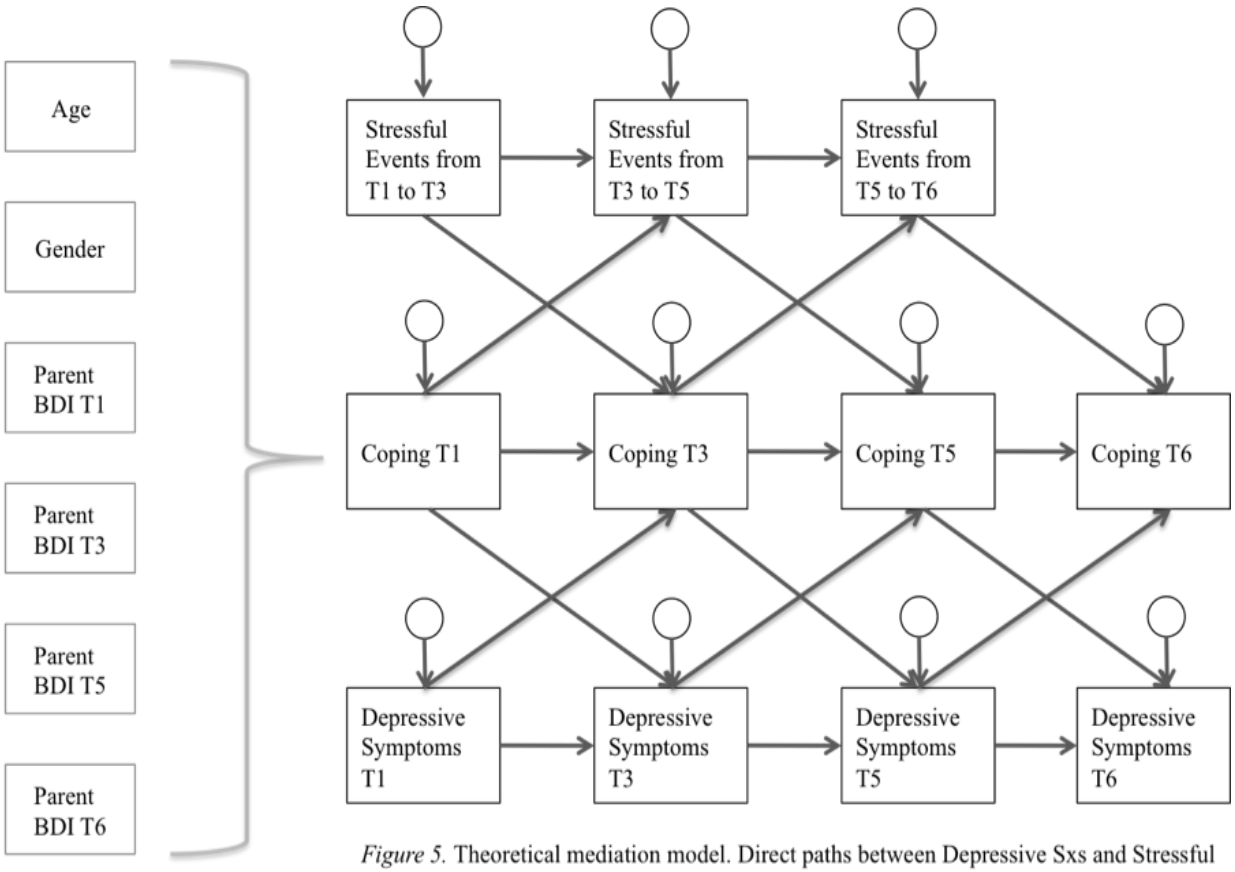


*Figure 5.* Theoretical mediation model. Direct paths between Depressive Sxs and Stressful Events, nonadjacent autoregressive paths, and covariances between errors within each time point were estimated but not depicted. Covariates included age, gender, and T1, T3, T5 and T6 Parent BDI (Beck Depression Inventory) scores which predicted all variables in model.

# CHAPTER IV

# DISCUSSION

This prospective study examined coping as a mediator of the relation between stressful life events and symptoms of depression in children and adolescents at four time points across approximately two years. Stressful events significantly predicted children's primary control and disengagement coping, which in turn predicted children's depressive symptoms. These findings contribute to the literature by providing prospective evidence that coping is a mediator of stress and depression in a sample that varied in risk.

Thus, one potential mechanism by which stressful life events may exert negative effects on children is through their using primary control coping strategies less and using less adaptive coping strategies such as disengagement more. These findings are consistent with the theoretical model of Compas (2006), which posits that the adverse effects of prolonged stress lead to impairments in the ability to cope. In turn, an inability to utilize adaptive coping strategies such as primary and secondary control coping is then a risk factor for internalizing problems (Compas, Connor-Smith et al., 2001).

The present study extends this line of research in two ways. First, previous findings have relied on cross-sectional data to test mediation. Empirical tests of mediation using a cross-sectional design, however, can be biased and potentially misleading (see Cole, & Maxwell, 2003). The present analyses utilized structural equation modeling to assess relations concurrently and across four time points over 22 months. Although some longitudinal studies have examined coping as a mediator in

samples of children of divorce (Sandler et al., 1994), and children exposed to marital conflict (Shelton, & Harold, 2007), the current study was the first multiple assessment, longitudinal investigation to examine three types of coping as possible mediators of the relation between stress and children's depressive symptoms in a sample at varied risk for mood disorder. We conducted a stringent test of the relations over time by using structural equation models that included autoregressive paths among stress, coping, and depressive symptoms at each time point. Additionally, children's age and gender, and parents' level of depressive symptoms were controlled at each time point.

Second, several of the studies examining coping as a mediator of the relation between stress and depressive symptoms were conducted with samples of offspring of depressed parents and assessed how children coped with the particular stressor of living with a depressed parent (Jaser et al., 2005; Langrock et al., 2002). These studies have found that secondary control coping mediated the relation between parental depression, an uncontrollable stressor, and children's depressive symptoms (Langrock et al., 2002; Jaser et al., 2005). The present study also used a sample of offspring of depressed (high risk) and nondepressed (low risk) parents, but assessed coping with regard to more general stressors, both controllable and uncontrollable, not just parental depression. In order to control for the varied risk status of the sample, parental depression was entered as a covariate. Thus, the present analyses allowed us to examine the relation between coping and depressive symptoms in the context of a broader array of stressors.

Correlation analyses revealed that stressful life events were positively correlated with disengagement coping, and negatively correlated with primary control coping and secondary control coping concurrently and at the next time points. Primary and secondary

control coping were negatively correlated with depressive symptoms, whereas disengagement coping was positively correlated with depressive symptoms concurrently and at the next time points. These associations replicate the basic descriptive research finding that children who experience higher levels of stress report lower levels of complex coping strategies such as problem-solving and cognitive restructuring (Valiente, et al., 2004). Additionally, patterns are consistent with previous findings that primary control and secondary control coping are associated with lower levels of internalizing symptoms. Results have been mixed regarding disengagement coping, with some finding disengagement to be associated with poorer outcomes (e.g., Compas et al., 2001; Connor-Smith et al., 2000; Wadsworth, & Compas, 2002), and others finding no significant relation between disengagement coping and adolescents' symptoms (Jaser et al., 2007). In the present analysis, disengagement coping was associated with higher levels of internalizing symptoms.

Examining the direct effects, we found that stressful life events predicted subsequent depressive symptoms in each model. This finding is consistent with prior research indicating a strong prospective association between stressful life events and levels of depressive symptoms (Hammen, & Goodman-Brown, 1990; Hilsman, & Garber, 1995; Grant, Compas, Thurm, McMahon, & Gipson, 2004; Kendler, Karkowski, & Prescott, 1999). Additionally, it has been found that children of depressed parents contribute to the number of stressful events they experience, perhaps due to maladaptive patterns of interpersonal interaction (Adrian, & Hammen, 1993; Hammen, Shih, & Brennan, 2004; Carter, Garber, Cieslo, & Cole, 2006). Results also provided some evidence for the direct effect of children's depressive symptoms on stressful life events.

Analyses of the indirect effects revealed that primary and disengagement stress responses partially mediated the relation between stressful events and depressive symptoms. Thus, coping responses may be one important pathways through which stressful events affect children's reactions to stress. The findings are consistent with previous longitudinal investigations, which reported that avoidance coping strategies mediated the relation between stress and depressive symptoms in children of divorce (Sandler et al., 1994) and maladaptive coping strategies mediated the relation between exposure to marital conflict and adjustment problems in children exposed to marital stress (Shelton, & Harold, 2007).

Regarding the direction of effects, previous analyses have found that symptoms were not a significant predictor of changes in coping over time (Wadsworth, & Berger, 2006; Sandler et al., 1994). This suggests that coping is not a proxy for symptoms; instead coping acts as a mediator of the prospective relation from stress to symptoms rather than from symptoms to stress. Results of the current study were consistent with this pattern. Although there were significant pathways from depressive symptoms to coping in the primary and secondary models, the direction of these effects was inconsistent, and only at T1 did coping predict fewer stressful events.

Findings regarding secondary control coping were not entirely consistent with investigations involving offspring of depressed parents. In the present analyses, secondary control coping did not significantly mediate the stress-depression relation. Studies have found evidence for secondary control coping as a mediator between stressor of parental depression and children's symptoms of psychopathology (Langrock et al., 2002). Several factors may explain these different findings. First, these prior studies of

offspring of depressed parents were cross-sectional. The current prospective analyses may have been a more stringent test of the mediation hypothesis. Second, secondary control coping strategies may have been endorsed less by children in the current sample. Alhough items on the COPE are strongly correlated with the primary control, secondary control, and disengagement coping domains used by Connor-Smith and colleagues (2000), a relatively smaller number of items on the COPE assessed secondary control coping than the other two domains. That is, the measure of coping used in the current study might not have assessed secondary control coping adequately.

Finally, previous investigations have focused on the uncontrollable stressor of parental depression, in particular. The current study assessed both controllable and uncontrollable stressors not specifically associated with parental depression. Better adjustment has been found when there is a good match between the stressor and the coping strategies used (Folkman, 1984; Forsythe, & Compas, 1987). Thus, secondary control coping might not have been a mediator due to the inclusion of controllable stressors in the model. It is still possible, however, that secondary control coping might be a mediator of the relation between *uncontrollable* stressors and child depressive symptoms.

Overall, the results of this study provide evidence of the longitudinal relation of stressful life events to the use of adaptive and cognitively demanding coping strategies, which in turn predicts depressive symptoms. A recent meta-analysis of the relations among six emotion regulation strategies and four types of psychopathology revealed that coping strategies such as rumination, suppression, problem-solving and avoidance were more strongly related to symptoms of psychopathology than strategies such as acceptance

and reappraisal (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Aldao and colleagues concluded that maladaptive strategies were more strongly related to psychopathology than adaptive strategies. The exception to this was that the absence of problem-solving may have a wide-ranging negative impact on well-being and contribute to the development of other maladaptive regulation strategies (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Thus, the absence of primary control response strategies, such as problem-solving, and the presence of such coping behaviors as avoidance and denial under conditions of stress are likely to lead to worse outcomes.

With regard to applications, cognitive load theory posits that high levels of stress may tax children's personal resources, which may then interfere with their ability to use effective coping strategies (Davies, & Cummings, 1994; Matthews, & Wells, 1996). Indeed, coping becomes less effective under conditions of greater stress (Connor-Smith et al., 2000; Jaser et al., 2005; Wadsworth, & Compas, 2002). Children exposed to high levels of stress are more likely to use coping strategies characterized by avoidance, denial, and wishful thinking. In turn, they are less likely to use more complex, goal-directed coping strategies such as problem-solving and seeking out support from others. Additionally, neurobiological research suggests that chronically elevated stress hormones can lead to the retraction of dendrites in the prefrontal cortex, the area of the brain associated with cognitively demanding cognitive processes (Wellman, 2001; McEwen, 2005). The current findings fit with the theoretical model that under conditions of elevated stress, complex strategies such as problem-solving are used less. There is a condition of double jeopardy in which stress both directly impacts current symptoms and at the same time also compromises a child's ability to cope with future stressors.

*Limitations*

Although this study advances the study of children's coping in the context of stressful life events, some limitations of the current study should be noted. First, the data for this study were based on questionnaires, which can be affected by rater bias, rather than independent observations of behavior that may provide a more objective assessment of stress response strategies. To address this issue, future studies should include composites of scores from multiple informants, perhaps using parent, sibling, and peers reports of a child's coping strategies as well as observations of behaviors in the context of stress.

Second, using a dispositional measure of coping (Carver et al., 1989) had both advantages and disadvantages. On the one hand, asking children how they cope in general allowed for the examination of coping strategies in response to both controllable and uncontrollable stressors. On the other hand, this measure required a child to recall and report how they coped across a variety of situations. Some aspects of coping are state-like and some are more stable and trait-like over time due to having to cope with the same chronic stressor (Wadsworth, & Berger, 2006). The efficacy of some coping responses may be context-specific (Jaser et al., 2007) and asking a child about how they cope in general may not adequately capture the specific interplay among stress, coping strategies, and depressive symptoms. Indeed, recent research has highlighted the importance of identifying how specific types of stressors predict specific types of internalizing and externalizing symptoms, rather than exploring various types of stress as broad risk factors for child and adolescent psychopathology (McMahon, Grant, Compas,

Thurm, & Ey, 2003). Future research examining coping as a mediator should use measures of coping that assesses how a child responds to multiple but specific, stressors.

Additionally, future studies should use prospective designs that incorporate more frequent waves of data collection and should closely examine the implications of the temporal spacing. Cole and Maxwell (2003) have shown that when testing for mediation it is important to measure the mediator and outcomes at optimal times. In the current study the duration between waves of data collection ranged from 4 months to 1 year. Findings have shown that recent stressors exert greater impact than more distal stressors on the mental health of children (Compas, Howell, Phares, Williams, & Ledoux, 1989). Following the recommendation of Compas (2004) to use measures of recent stressors as predictors, the current study assessed stressful life events that occurred up to the time point at which coping strategies and depressive symptoms were measured. Thus, there were shorter intervals for the predictive paths from stressful life events to coping than from depressive symptoms to coping. It is possible that important effects from depressive symptoms to coping might occur over shorter intervals. As an independent variable becomes more distal, the influence of random factors becomes increasingly likely (Shrout, & Bolger, 2002). A more time-sensitive design might have allowed for a closer examination of the impact of coping strategies on levels of depressive symptoms. Nevertheless, our ability to detect an association between depressive symptoms and coping over long intervals suggests that these findings are likely robust.

Finally, using a sample that varied in risk for depression had advantages and disadvantages. On the one hand, this type of sample increased the range of scores on measures of stressful life events, coping strategies, and depressive symptoms, thereby

increasing power to detect effects. On the other hand, these findings might not generalize to a purely normative sample.

In conclusion, results from the present study highlight the importance of examining the longitudinal, meditational chain positioning stressful life events as predictive of coping strategies and coping strategies as predictive of depressive symptoms. Previous research has shown that prolonged stress has a significant adverse effect on the brain, specifically those regions responsible for higher order executive functions that regulate successful adaptation to stress (McEwen, 1998; McEwen, 2005). The present study showed that stressful life events might have long-term effects on children through their coping behaviors over and above the influence of existing psychological problems. These findings can inform the development of targeted interventions aimed at decreasing maladaptive coping strategies such as disengagement and increasing strategies that have the most significant impact on well-being in children experiencing high levels of stress, such as primary coping. Future studies should explore the developmental trajectories of coping, especially in the context of specific types of stressors. Such research is needed to better understand the normative development of coping skills as well as how coping profiles emerge as a long-term effect of different combinations of stressors. Identifying when developmental shifts in coping occur and how those differ for children experiencing chronic stress can help researchers and clinicians target individuals who are at greatest risk for negative outcomes.

Table 1. Demographic Characteristics and Parents' Baseline Depressive Symptom Scores

| **CHILDREN** | **High-Risk N = 129** | **Low-Risk N=98** |
|---|---|---|
| **Age** [Mean (*SD*)] | 11.96 (2.40) | 12.33 (2.19) |
| **Girls** [N (%)] | 68 (52.7%) | 54 (55.1) |
| **Ethnicity** [N (%)] | | |
| White, non-Hispanic | 90 (69.8%) | 68 (69.4%) |
| African-American | 27 (20.9%) | 22 (22.4%) |
| Asian | 2 (1.5%) | 1 (1.0%) |
| Multi-racial | 10 (7.8%) | 6 (6.1%) |
| **PARENTS** | **Depressed N=129** | **Nondepressed N=98** |
| **Age** [Mean (*SD*)] | 41.32 (6.8) | 44.48 (5.0) |
| **Female** [N (%)] | 95 (73.5%) | 77 (77.5%) |
| **Parent Education** [Mean (*SD*)] | 14.71 (14.71) | 15.48 (15.45) |
| **BDI-II** [Mean (*SD*)] | 25.77 (11.69) | 1.91 (2.48) |

SD = Standard Deviation; BDI-II = Beck Depression Inventory, second edition

Table 2. Means, Standard Deviations and Correlations of Study Variables

| | Mean | SD | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. T1 LEQ | 8.07 | 5.52 | 1 | | | | | | | | | |
| 2. T1 CDI | 6.51 | 5.83 | .44*** | 1 | | | | | | | | |
| 3. T1 Primary Control Coping | .509 | .048 | -.13 | -.39*** | 1 | | | | | | | |
| 4. T1 Secondary Control Coping | .257 | .035 | -.16* | -.19** | - | 1 | | | | | | |
| 5. T1 Disengagement Coping | .232 | .056 | .20** | .43*** | - | - | 1 | | | | | |
| 6. T3 LEQ | 4.10 | 4.15 | .62*** | .40*** | -.06 | -.15* | .14 | 1 | | | | |
| 7. T3 CDI | 4.09 | 4.92 | .35*** | .62*** | -.35*** | -.19** | .42*** | .52*** | 1 | | | |
| 8. T3 Primary Control Coping | .513 | .053 | -.03 | -.26*** | .47*** | -.02 | -.40*** | -.03 | -.22** | 1 | | |
| 9. T3 Secondary Control Coping | .26 | .039 | -.2** | -.29*** | .04 | .54*** | -.34*** | -.21** | -.24** | - | 1 | |
| 10. T3 Disengagement Coping | .226 | .055 | .16* | .40*** | -.46*** | -.34*** | .60*** | .18* | .38*** | - | - | 1 |
| 11. T5 LEQ | 3.36 | 3.39 | .56*** | .45*** | -.29*** | -.15* | .32*** | .57*** | .33*** | -.22** | -.14 | .30*** |
| 12. T5 CDI | 4.06 | 5.17 | .45*** | .53*** | -.32*** | -.12 | .33*** | .47*** | .45*** | -.16* | -.25** | .30*** |
| 13. T5 Primary Control Coping | .524 | .051 | -.10 | -.19** | .35*** | .13 | -.38*** | -.12 | -.08 | .42*** | .08 | -.42*** |
| 14. T5 Secondary Control Coping | .264 | .037 | -.18* | -.08 | -.01 | .36*** | -.21** | -.18* | -.15* | .01 | .52*** | -.36*** |
| 15. T5 Disengagement Coping | .213 | .056 | .21** | .23** | -.32*** | -.37*** | .50*** | .23*** | .18* | -.39*** | -.42*** | .63*** |
| 16. T6 LEQ | 3.64 | 3.35 | .53*** | .35*** | -.16* | -.04 | .16* | .49*** | .34*** | -.05 | -.14 | .13 |
| 17. T6 CDI | 4.08 | 5.34 | .36*** | .51*** | -.23** | -.15 | .28*** | .46*** | .48*** | -.22** | -.14 | .30*** |
| 18. T6 Primary Control Coping | .514 | .057 | -.06 | -.18* | .40*** | .20* | -.47*** | -.06 | -.12 | .36*** | .18* | -.45*** |
| 19. T6 Secondary Control Coping | .272 | .039 | -.17* | -.22** | -.07 | .30*** | -.12 | -.19* | -.19* | .06 | .45*** | -.36*** |
| 20. T6 Disengagement Coping | .212 | .059 | .12 | .33*** | -.37*** | -.36*** | .54*** | .14 | .24** | -.36*** | -.44*** | .63*** |

Table 2 (Continued).

| | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 11. T5 LEQ | 1 | | | | | | | | | | |
| 12. T5 CDI | .51*** | 1 | | | | | | | | | |
| 13. T5 Primary Control Coping | -.24*** | -.30*** | 1 | | | | | | | | |
| 14. T5 Secondary Control Coping | -.12 | -.21** | - | 1 | | | | | | | |
| 15. T5 Disengagement Coping | .30*** | .41*** | - | - | 1 | | | | | | |
| 16. T6 LEQ | .63*** | .45*** | -.14 | -.09 | .19* | 1 | | | | | |
| 17. T6 CDI | .39*** | .62*** | -.31*** | -.21** | .43*** | .40*** | 1 | | | | |
| 18. T6 Primary Control Coping | -.23** | -.35*** | .62*** | -.03 | -.55*** | -.14 | -.37*** | 1 | | | |
| 19. T6 Secondary Control Coping | -.04 | .20* | -.04 | .57*** | -.35*** | -.08 | -.27*** | - | 1 | | |
| 20. T6 Disengagement Coping | .22** | .41*** | -.53*** | -.33*** | .72*** | .14 | .49*** | - | - | 1 | |

*p<.05; **p<.01; ***p<.001

SD = Standard Deviation; T = Time point; LEQ = Life Events Questionnaire; CDI = Children's Depression Inventory

Table 3.

Direct and Cross-Lagged Paths for Primary Control Coping Model

| **Direct Effects** (Between Stress and Depressive Sxs) | ***b*** | **SE** | ***t*** | **p-value** | ***β*** |
|---|---|---|---|---|---|
| T1-T3 Stressful Events → T3 Depressive Sxs | .496 | .063 | 7.876 | < 0.001 | .406 |
| T1 Depressive Sxs → T3-T5 Stressful Events | .113 | .039 | 2.917 | .004 | .196 |
| T1-T3 Stressful Events → T5 Depressive Sxs | .169 | .098 | 1.725 | .085 | .138 |
| T3-T5 Stressful Events → T5 Depressive Sxs | .426 | .111 | 3.842 | < 0.001 | .281 |
| T3 Depressive Sxs → T5-T6 Stressful Events | .04 | .048 | .823 | .41 | .06 |
| T3-T5 Stressful Events → T6 Depressive Sxs | -.138 | .119 | -1.154 | .249 | -.086 |
| T5-T6 Stressful Events → T6 Depressive Sxs | .189 | .112 | 1.689 | .091 | .119 |
| **Cross-Lagged Paths** (Between Primary Coping and Stress) | ***b*** | **SE** | ***t*** | **p-value** | ***β*** |
| T1 Primary Coping → T3-T5 Stressful Events | -9.54 | 4.234 | -2.254 | .024 | -.138 |
| T1-T3 Stressful Events → T3 Primary Coping | .0001 | .001 | .318 | .75 | .023 |
| T3 Primary Coping → T5-T6 Stressful Events | 6.701 | 3.8 | 1.763 | .078 | .104 |
| T3-T5 Stressful Events → T5 Primary Coping | -.003 | .001 | -2.69 | .007 | -.196 |
| T5-T6 Stressful Events → T6 Primary Coping | -.001 | .001 | -.785 | .433 | -.054 |
| **Cross-Lagged Paths** (Between Primary Coping and Depressive Sxs) | ***b*** | **SE** | ***t*** | **p-value** | ***β*** |
| T1 Primary Coping → T3 Depressive Sxs | -16.891 | 5.191 | -3.254 | .001 | -.162 |
| T1 Depressive Sxs → T3 Primary Coping | -.001 | .001 | -1.446 | .148 | -.111 |
| T3 Primary Coping → T5 Depressive Sxs | 2.908 | 5.87 | .496 | .62 | .03 |
| T3 Depressive Sxs → T5 Primary Coping | .002 | .001 | 2.342 | .019 | .173 |
| T5 Primary Coping → T6 Depressive Sxs | -16.168 | 5.893 | -2.744 | .006 | -.153 |
| T5 Depressive Sxs → T6 Primary Coping | -.002 | .001 | -2.924 | .003 | -.198 |
| **Error Variance** | **Estimate** | **SE** | ***t*** | **p-value** | ***r*** |
| T1-T3 Stressful Events | 14.672 | 1.429 | 10.269 | < 0.001 | - |
| T3-T5 Stressful Events | 6.328 | .644 | 9.829 | < 0.001 | - |
| T5-T6 Stressful Events | 5.91 | .618 | 9.56 | < 0.001 | - |
| T1 Primary Coping | .002 | .000 | 10.002 | < 0.001 | - |
| T3 Primary Coping | .002 | .000 | 9.96 | < 0.001 | - |
| T5 Primary Coping | .002 | .000 | 9.808 | < 0.001 | - |

| | | | | | |
|---|---|---|---|---|---|
| T6 Primary Coping | .002 | .000 | 9.044 | < 0.001 | - |
| T1 Depressive Sxs | 30.284 | 2.875 | 10.532 | < 0.001 | - |
| T3 Depressive Sxs | 10.039 | 1.001 | 10.028 | < 0.001 | - |
| T5 Depressive Sxs | 14.938 | 1.514 | 9.868 | < 0.001 | - |
| T6 Depressive Sxs | 13.916 | 1.462 | 9.519 | < 0.001 | - |
| **Error Covariances** | **Estimate** | **SE** | ***t*** | **p-value** | ***r*** |
| T1 Primary Coping – T1-T3 Stressful Events | -.016 | .013 | -1.175 | .24 | -.084 |
| T1 Depressive Sxs – T1 Primary Coping | -.105 | .02 | -5.259 | < 0.001 | -.393 |
| T1 Depressive Sxs – T1-T3 Stressful Events | 7.968 | 1.539 | 5.176 | < 0.001 | .378 |
| T3 Primary Coping – T3-T5 Stressful Events | -.006 | .008 | -.763 | .445 | -.056 |
| T3 Depressive Sxs – T3 Primary Coping | -.015 | .01 | -1.485 | .138 | -.107 |
| T3 Depressive Sxs – T3-T5 Stressful Events | -1.46 | .588 | -2.484 | .013 | -.183 |
| T5 Depressive Sxs – T5 Primary Coping | -.038 | .013 | -3.013 | .003 | -.223 |
| T5 Primary Coping – T5-T6 Stressful Events | -.004 | .008 | -.498 | .618 | -.038 |
| T5 Depressive Sxs – T5-T6 Stressful Events | .983 | .711 | 1.382 | .167 | .105 |
| T6 Primary Coping – T6 Depressive Sxs-.105 | -.034 | .013 | -2.688 | .007 | -.215 |

Table 4. Direct and Cross-Lagged Paths for Secondary Control Coping Model

| **Direct Effects** (Between Stress and Depressive Sxs) | ***b*** | **SE** | ***t*** | **p-value** | ***β*** |
|---|---|---|---|---|---|
| T1-T3 Stressful Events → T3 Depressive Sxs | .474 | .064 | 7.385 | < 0.001 | .389 |
| T1 Depressive Sxs → T3-T5 Stressful Events | .145 | .036 | 4.018 | < 0.001 | .25 |
| T1-T3 Stressful Events → T5 Depressive Sxs | .194 | .097 | 1.999 | .046 | .158 |
| T3-T5 Stressful Events → T5 Depressive Sxs | .395 | .109 | 3.606 | < 0.001 | .26 |
| T3 Depressive Sxs → T5-T6 Stressful Events | .021 | .047 | .457 | .648 | .032 |
| T3-T5 Stressful Events → T6 Depressive Sxs | -.066 | .121 | -.546 | .585 | -.042 |
| T5-T6 Stressful Events → T6 Depressive Sxs | .159 | .114 | 1.39 | .165 | .10 |
| **Cross-Lagged Paths** (Between Secondary Coping and Stress) | ***b*** | **SE** | ***t*** | **p-value** | ***β*** |
| T1 Secondary Coping → T3-T5 Stressful Events | -8.028 | 5.704 | -1.407 | .159 | -.083 |
| T1-T3 Stressful Events → T3 Secondary Coping | .000 | .001 | -.473 | .636 | -.032 |
| T3 Secondary Coping → T5-T6 Stressful Events | -1.675 | 5.136 | -.326 | .744 | -.019 |
| T3-T5 Stressful Events → T5 Secondary Coping | .000 | .001 | .077 | .939 | .005 |
| T5-T6 Stressful Events → T6 Secondary Coping | .000 | .001 | .134 | .894 | .01 |
| **Cross-Lagged Paths** (Between Secondary Coping and Depressive Sxs) | ***b*** | **SE** | ***t*** | **p-value** | ***β*** |
| T1 Secondary Coping → T3 Depressive Sxs | -6.652 | 7.101 | -.937 | .349 | -.046 |
| T1 Depressive Sxs → T3 Secondary Coping | -.001 | .000 | -2.711 | .007 | -.178 |
| T3 Secondary Coping → T5 Depressive Sxs | -11.883 | 7.93 | -1.499 | .134 | -.089 |
| T3 Depressive Sxs → T5 Secondary Coping | .000 | .001 | -.441 | .659 | -.031 |
| T5 Secondary Coping → T6 Depressive Sxs | -2.872 | 8.148 | -.352 | .724 | -.02 |
| T5 Depressive Sxs → T6 Secondary Coping | .000 | .001 | -.508 | .612 | -.036 |
| **Error Variance** | **Estimate** | **SE** | ***t*** | **p-value** | ***r*** |
| T1-T3 Stressful Events | 14.675 | 1.429 | 10.266 | < 0.001 | - |
| T3-T5 Stressful Events | 6.417 | .652 | 9.838 | < 0.001 | - |
| T5-T6 Stressful Events | 5.987 | .625 | 9.578 | < 0.001 | - |
| T1 Secondary Coping | .001 | .000 | 9.975 | < 0.001 | - |
| T3 Secondary Coping | .001 | .000 | 9.912 | < 0.001 | - |
| T5 Secondary Coping | .001 | .000 | 9.788 | < 0.001 | - |

| | | | | | |
|---|---|---|---|---|---|
| T6 Secondary Coping | .001 | .000 | 9.082 | < 0.001 | - |
| T1 Depressive Sxs | 30.446 | 2.891 | 10.532 | < 0.001 | - |
| T3 Depressive Sxs | 10.491 | 1.041 | 10.078 | < 0.001 | - |
| T5 Depressive Sxs | 14.708 | 1.492 | 9.86 | < 0.001 | - |
| T6 Depressive Sxs | 14.385 | 1.508 | 9.537 | < 0.001 | - |
| **Error Covariances** | **Estimate** | **SE** | ***t*** | **p-value** | ***r*** |
| T1 Secondary Coping – T1-T3 Stressful Events | -.015 | .009 | -1.625 | .104 | -.117 |
| T1 Depressive Sxs – T1 Secondary Coping | -.033 | .013 | -2.474 | .013 | -.177 |
| T1 Depressive Sxs – T1-T3 Stressful Events | 7.994 | 1.544 | 5.177 | < 0.001 | .378 |
| T3 Secondary Coping – T3-T5 Stressful Events | .008 | .006 | 1.303 | .193 | .097 |
| T3 Depressive Sxs – T3 Secondary Coping | -.001 | .007 | -.155 | .877 | -.011 |
| T3 Depressive Sxs – T3-T5 Stressful Events | -1.225 | .603 | -2.036 | .042 | -.149 |
| T5 Depressive Sxs – T5 Secondary Coping | -.017 | .009 | -1.915 | .055 | -.141 |
| T5 Secondary Coping – T5-T6 Stressful Events | -.001 | .006 | -.108 | .914 | -.008 |
| T5 Depressive Sxs – T5-T6 Stressful Events | .965 | .71 | 1.358 | .175 | .103 |
| T6 Secondary Coping – T6 Depressive Sxs | -.012 | .009 | -1.296 | .195 | -.102 |

Table 5. Direct and Cross-Lagged Paths for Disengagement Coping Model

| **Direct Effects** (Between Stress and Depressive Sxs) | ***b*** | **SE** | ***t*** | **p-value** | ***β*** |
|---|---|---|---|---|---|
| T1-T3 Stressful Events → T3 Depressive Sxs | .488 | .063 | 7.806 | < 0.001 | .397 |
| T1 Depressive Sxs → T3-T5 Stressful Events | .108 | .038 | 2.804 | .005 | .186 |
| T1-T3 Stressful Events → T5 Depressive Sxs | .107 | .093 | 1.151 | .25 | .087 |
| T3-T5 Stressful Events → T5 Depressive Sxs | .428 | .11 | 3.908 | < 0.001 | .282 |
| T3 Depressive Sxs → T5-T6 Stressful Events | .063 | .05 | 1.263 | .207 | .096 |
| T3-T5 Stressful Events → T6 Depressive Sxs | -.086 | .116 | -.743 | .458 | -.054 |
| T5-T6 Stressful Events → T6 Depressive Sxs | .166 | .111 | 1.488 | .137 | .104 |
| **Cross-Lagged Paths** (Between Disengagement Coping and Stress) | ***b*** | **SE** | ***t*** | **p-value** | ***β*** |
| T1 Disengagement Coping → T3-T5 Stressful Events | 10.057 | 3.678 | 2.735 | .006 | .168 |
| T1-T3 Stressful Events → T3 Disengagement Coping | .000 | .001 | .414 | .679 | .027 |
| T3 Disengagement Coping → T5-T6 Stressful Events | -5.466 | 3.849 | -1.42 | .156 | -.09 |
| T3-T5 Stressful Events → T5 Disengagement Coping | .002 | .001 | 2.118 | .034 | .133 |
| T5-T6 Stressful Events → T6 Disengagement Coping | .001 | .001 | .834 | .404 | .049 |
| **Cross-Lagged Paths** (Between Disengagement Coping and Depressive Sxs) | ***b*** | **SE** | ***t*** | **p-value** | ***β*** |
| T1 Disengagement Coping → T3 Depressive Sxs | 17.248 | 4.515 | 3.82 | < 0.001 | .19 |
| T1 Depressive Sxs → T3 Disengagement Coping | .002 | .001 | 2.766 | .006 | .187 |
| T3 Disengagement Coping → T5 Depressive Sxs | 2.053 | 5.954 | .345 | .73 | .022 |
| T3 Depressive Sxs → T5 Disengagement Coping | -.002 | .001 | -2.933 | .003 | -.191 |
| T5 Disengagement Coping → T6 Depressive Sxs | 16.503 | 5.686 | 2.902 | .004 | .172 |
| T5 Depressive Sxs → T6 Disengagement Coping | .001 | .001 | 1.954 | .05 | .12 |
| **Error Variance** | **Estimate** | **SE** | ***t*** | **p-value** | ***r*** |
| T1-T3 Stressful Events | 14.684 | 1.43 | 10.271 | < 0.001 | - |
| T3-T5 Stressful Events | 6.227 | .634 | 9.819 | < 0.001 | - |
| T5-T6 Stressful Events | 5.931 | .62 | 9.567 | < 0.001 | - |
| T1 Disengagement Coping | .003 | .000 | 10.024 | < 0.001 | - |
| T3 Disengagement Coping | .002 | .000 | 9.906 | < 0.001 | - |
| T5 Disengagement Coping | .002 | .000 | 9.722 | < 0.001 | - |
| T6 Disengagement Coping | .001 | .000 | 8.977 | < 0.001 | - |

| | | | | | |
|---|---|---|---|---|---|
| T1 Depressive Sxs | 30.615 | 2.907 | 10.533 | < 0.001 | - |
| T3 Depressive Sxs | 9.916 | .989 | 10.025 | < 0.001 | - |
| T5 Depressive Sxs | 14.896 | 1.509 | 9.87 | < 0.001 | - |
| T6 Depressive Sxs | 13.9 | 1.458 | 9.537 | < 0.001 | - |
| **Error Covariances** | **Estimate** | **SE** | ***t*** | **p-value** | ***r*** |
| T1 Disengagement Coping – T1-T3 Stressful Events | .032 | .015 | 2.083 | .037 | .149 |
| T1 Depressive Sxs – T1 Disengagement Coping | .127 | .023 | 5.522 | < 0.001 | .414 |
| T1 Depressive Sxs – T1-T3 Stressful Events | 8.095 | 1.55 | 5.222 | < 0.001 | .382 |
| T3 Disengagement Coping – T3-T5 Stressful Events | .001 | .008 | .116 | .908 | .009 |
| T3 Depressive Sxs – T3 Disengagement Coping | .018 | .01 | 1.88 | .06 | .136 |
| T3 Depressive Sxs – T3-T5 Stressful Events | -1.54 | .581 | -2.65 | .008 | -.196 |
| T5 Depressive Sxs – T5 Disengagement Coping | .059 | .012 | 4.79 | < 0.001 | .371 |
| T5 Disengagement Coping – T5-T6 Stressful Events | .009 | .008 | 1.127 | .26 | .086 |
| T5 Depressive Sxs – T5-T6 Stressful Events | .998 | .711 | 1.403 | .16 | .106 |
| T6 Disengagement Coping – T6 Depressive Sxs | .043 | .012 | 3.672 | < 0.001 | .30 |

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